CASE REPORT

The Lazarus phenomenon following recreational drug use

A Walker, H McClelland, J Brenchley

Abstract

A case is reported of the Lazarus phenomenon (the return of spontaneous circulation after cardiopulmonary resuscitation had been abandoned) in a patient following recreational drug use. The implications for management of cardiac arrest in the emergency department are discussed.
(*Emerg Med J* 2001;**18**:74–75)

Keywords: Lazarus phenomenon; recreational drug use

The unexpected return of spontaneous circulation (ROSC) after resuscitation has been abandoned is uncommon and was first described by Linko *et al*[1] in 1982. The term "Lazarus phenomenon" was coined by Bray[2] to describe this situation after the biblical character Lazarus who Jesus reputedly raised to life.[3] There have been more than 25 cases reported in the literature but it remains unfamiliar to emergency department staff. Various conditions have been associated with the phenomenon, however there has not previously been any case reported in a patient following recreational drug use.[4]

A case of Lazarus phenomenon associated with a drug overdose seen in the emergency department is presented.

Case report

Paramedics were called to a 27 year old man who was allegedly a heroin and ecstasy user who had collapsed after an intravenous injection of drugs. On arrival the crew found that his GCS was 3/15 and his respiratory rate 4 per minute. He had two apparently new puncture wounds in his left antecubital fossa. He was given intramuscular naloxolone 800 μg as peripheral intravenous access could not be obtained, followed by another 400 μg intramuscularly. The patient then recovered and walked to the ambulance. There was no significant past medical history.

In transit, the patient's respiratory rate decreased and he had a cardiorespiratory arrest as the ambulance arrived at the hospital.

Basic life support (BLS) was started and the patient was transferred to the resuscitation room.

On arrival he was in asystole and BLS was continued. The resuscitation team comprised an accident and emergency (A&E) specialist registrar (team leader) and two senior A&E nurses who were all familiar with advanced life support guidelines. The patient was cannulated (external jugular vein) and intubated, and the resuscitation clock was started. The venous blood glucose was within normal range. Over the next 25 minutes several asystole cycles were completed and the patient was given intravenously: adrenaline (epinephrine) 1 mg, 1 mg, 1 mg, 5 mg; atropine 3 mg; naloxone 800 μg × 3, 1200 μg × 1; sodium bicarbonate 8.4% 50 ml.

During resuscitation the ECG monitor detected the chest compressions and showed asystole at each reassessment with no central pulse detectable.

The decision to discontinue resuscitation was taken after 25 minutes of timed resuscitation when the patient was still asystolic and pulseless with no respiratory effort. The time of death was noted verbally.

The team leader was then called to see another patient and about a minute later the A&E sister noted a rhythm on the monitor and a palpable radial pulse. The first systolic blood pressure recorded was 140. Assisted ventilation was restarted and the patient was transferred to the intentsive care unit.

After 13 days in the intensive care unit with aspiration pneumonia, septicaemia and ARDS, he was transferred to a general ward. He was discharged home 18 days after admission with full neurological recovery.

The qualitative urinary toxicology screen detected opioids and cocaine, and the patient denied taking any other drugs. No significant level of alcohol was detected.

Discussion

Previous reports of this phenomenon have mostly been related to patients with ischaemic events or associated with exacerbations of chronic disease.

We have been unable to find any reports in the literature of this phenomenon related to drug overdose.

Although no single cause has been identified, there have been several mechanisms of survival postulated for Lazarus phenomenon patients. Hyperkalaemia associated with renal failure can cause cardiac arrest, and in one such case survival has been documented, with

Accident and Emergency Department, Leeds General Infirmary, Great George Street, Leeds LS1 3EX, UK

Correspondence to:
Dr Walker, Specialist Registrar
(alison.walkerl@virgin.net)

Accepted 28 June 2000

Emerg Med J: first published as 10.1136/emj.18.1.74 on 1 January 2001. Downloaded from http://emj.bmj.com/ on October 20, 2021 by guest. Protected by copyright.

appropriate treatment, after 26 minutes of asystole.[5] Increased intrathoracic pressure with decreased venous return has also been associated with ROSC after cessation of cardiopulmonary resuscitation (CPR), most commonly in patients with known COPD or asthma, or after mechanical ventilation.[6–8] High dose adrenaline (epinephrine) has also been thought to contribute to unexpected ROSC after CPR has been abandoned. In one case of EMD arrest high dose adrenaline (5 mg) was given, then resuscitation stopped three minutes later.[9] After a further two minutes the patient was noted to be making respiratory effort and a pulse was palpable, however the patient survived for less than one hour. The author attributed this to the fact that adrenaline delivery to the heart was delayed. This may have been attributable to placement of the cannula in the antecubital fossa.

In the case presented none of the above theories seemed to be relevant. The first potassium measurement recorded was 4.8 mmol/l. The patient had no underlying airway disease, and although he had aspirated stomach contents before intubation there was little airway resistance to ventilation during resuscitation. All drugs were given via a size 14 gauge cannula in the right external jugular vein, which although technically was not a central line, allows more rapid delivery of drugs to the coronary circulation than use of other peripheral veins.

This case is apparently unique in the literature as the patient was young with no known underlying disease and a clear history of recreational drug use. Opioids are known to be cardiovascular and respiratory depressants. Cocaine is a stimulant but has been reported as contributing to cases of cardiac arrhythmias and respiratory arrest.[10]

Recommendations from the literature suggest evaluation of vital signs for 5 to 10 minutes after discontinuation of resuscitation and before certification of death.[1 6] This simple solution may avoid missing similar cases. Clearly this may present difficulties in the emergency department management of the patients particularly if relative are present witnessing resuscitation. Further discussion is needed to clarify these issues.

Contributors
Alison Walker and Heather McClelland initiated the original idea for the paper. Alison Walker and Jane Brenchley carried out the literature search. All the authors contributed to writing the paper.

1 Linko K, Honkavaara P, Salmenpera M. recovery after discontinued cardiopulmonary resuscitation. *Lancet* 1982;**13**:261–3.
2 Bray JG. The Lazarus phenomen revisited. *Anesthesiology* 1993; **341**:841.
3 *Holy Bible*. Gospel of St John xix, 38–44. (New International version). London: Hodder and Stoughton, 1993,.
4 Maleck WH, Piper SN, Triem J, *et al*. Unexpected return of spontaneous circulation after cessation of resuscitation (Lazarus phenomenon). *Resuscitation* 1998;**39**:125–8.
5 Quick G, Bastani B. Prolonged asystolic hyperkalemic cardiac arrest with no neurologic sequelae. *Ann Emerg Med* 1994;**24**:305–11.
6 Martens P, Vandekerckhove Y, Mullie A. Restoration of spontaneous circulation after cessation of cardiopulmonary resuscitation. [Letter]. *Lancet* 1993;**341**:841.
7 Koblin D. Prolonged asystolic hyperkalemic cardiac arrest with no neurological sequelae: the Lazarus Phenomenon arises again. [Letter]. *Ann Emerg Med* 1995;**25**:562–3.
8 Koblin D. The Lazarus phenomenon revisited: I. [Letter]. *Anesthesiology* 1993;**79**:1438.
9 Bradbury N. Lazarus phenomenon: another case? *Resuscitation* 1999;**41**:87.
10 Hassan TB, Pickett JA, Durham S, *et al*. Diagnostic indicators in the early recognition of severe cocaine intoxication. *J Accid Emerg Med* 1996;**13**:261–3.

Emerg Med J: first published as 10.1136/emj.18.1.74 on 1 January 2001. Downloaded from http://emj.bmj.com/ on October 20, 2021 by guest. Protected by copyright.